NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, January 2, 1883.

Dr. LEONARD WEBER read a paper entitled "A case of syphiloma of the right vertebral, with thrombosis of the basilar artery," presenting at the same time the specimen showing the occlusion.

The patient, æt. forty-two, single, was seen in 1878. For a few weeks previous to his visit, he had felt unusually tired and weak, was losing his appetite, had occasional nausea, irregular action of the bowels, and frequent headaches of the nature of painful pressure on top of head, and disturbed sleep. Twelve days later he had vertigo and a constant roaring noise in both his ears; numbness of right leg and the pressure on top of head had increased; also paresis of the right upper and lower extremities, and of the right half of the face and tongue. Eye-sight normal; no strabismus. Articulation and expression undisturbed. He was able to void his urine, and his bowels were evacuated by a simple enema. Vertigo continued, also pressure on top of head; no pain anywhere else, no muscular throbbings.

On questioning him with regard to syphilis, he admitted having had a small sore on his penis twelve years ago. In the morning of the thirteenth day he was taken with a terrible fit of general convulsions, lasting several minutes. While this paroxysm lasted, cyanosis developed and became so intense that his face became almost black. He lost consciousness after the first attack, the convulsions returned about every half hour subsequently, and he died in deep coma the same morning. The autopsy was made the following morning, and

limited to the head. After removing the brain the basilar artery was found to be filled completely with a firm clot, reaching from the place of union of the two vertebrals about an inch upward. On making a longitudinal section, we cut into a dense little tumor growing from the inner walls of the right vertebral, just at the junction with its fellow, and almost completely obliterating the lumen of the basilar artery at the very beginning of its course. A microscopic examination showed the tumor to be composed of small cells and connective tissue, corresponding in its character to that of the similar gummatous neoplasms of the cerebral arteries as described by Heubner and others.

We further noticed a small but sharp exostosis in the left fossa occipitalis. The crista galli was found to be unusually long, sending a number of osseous stalactites into the falciform process of the dura mater. In the substance of the brain nothing abnormal was found.

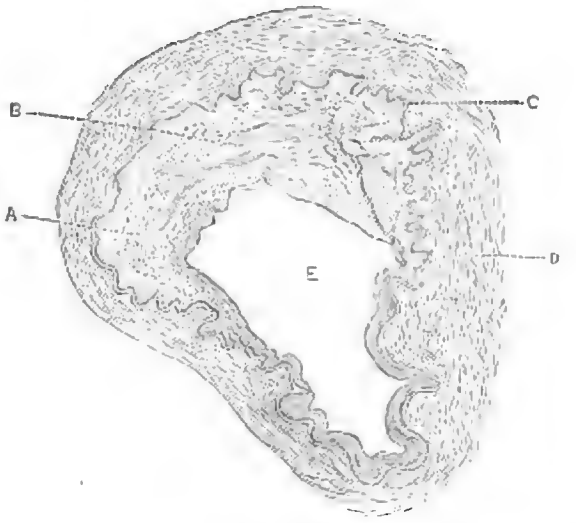
In examining the specimen of the basilar with portions of the two vertebral arteries, it will be seen that the thrombus extends from the junction of both vertebrals, an inch or so upward, depriving the medulla oblongata of the supply of arterial blood and causing the death of the patient.

Dr. JOHN A. WYETH corroborated Dr. Weber's account, and related a second case in which the autopsy had shown a very similar lesion.

The patient was a female, æt. about sixty-five. Syphilitic history dating back twenty years. For about fifteen years she had had complete hemiplegia of the left side, partial of the right, and incontinence of fæces and urine. When first seen by Dr. Wyeth, five years ago, articulation was indistinct, but mind was unimpaired. She refused all treatment, and died suddenly in the night, in 1882.

The skull was about one third thicker than usual, and presented a few ostoses. Brain in fair condition. Cerebral arteries permeable, but in a condition of fatty degeneration. Basilar almost occluded by endarteritis obliterans.

The patient died from anæmia of the medulla. The following cut illustrates the nature of the lesion.



E. Lumen, about two thirds obliterated. D. The muscular coat and adventitia nodulated by inflammatory changes. C. The elastic lamina. A. Inflammatory new-formed tissue in the intima, composed of large spindle and round (on section) cells, with the normal cells of the intima next to the elastic lamina and the lumen of the vessel. B. The hyaline substance seen distinctly with a higher power.

After a brief discussion upon the eccentricity of syphilitic lesions and their symptoms, the Society listened to the reading of the paper of the evening upon Allochiria by Dr. WILLIAM A. HAMMOND.

ALLOCHIRIA: ITS NATURE AND SEAT.

On the 4th of November, 1882, I examined, in conjunction with Dr. L. A. Stimson, and at the request of the Corporation Counsel, a gentleman who, it was asserted, had received a serious injury of the spine. It appeared that on the 27th of February, 1881, he was driving from the city to his residence at South Yonkers. It was a dark, foggy, and rainy night, and he drove into a ditch which the ice and snow had formed entirely across the road. The front axle of his carriage was at once broken, and he was jerked forward against the dash-board. The horses started to run, and dragged the vehicle, with him in the constrained position mentioned, for the distance of about two hundred feet

before they were stopped. He then, not thinking himself to be severely injured, procured another carriage, and drove on, in his wet clothes, through the rain to his home, which he reached at about half-past two o'clock the following morning. In a day or two, symptoms indicating spinal trouble began to be developed. He some time afterward consulted a physician of this city, who diagnosticated Pott's disease. This opinion was confirmed by a surgeon to whom the physician took him, and a plaster jacket was applied. Amendment soon began, and, finding the jacket uncomfortable, he removed it; but, his symptoms recurring, it was replaced, and, in addition, an apparatus designed to keep the head from resting directly on the vertebral column was applied.

Several months elapsed, during which he was at times better and at others worse. Upon the whole, however, there was no decided improvement. The fact that he had brought an action for heavy damages against the city was the immediate cause of my examination.

So far as I could determine from the clinical history given me by the patient, I was satisfied that at no time had he suffered from injury of the vertebral column or subsequent Pott's disease. Certainly he exhibited no symptoms of that affection when I visited him, and the surgeon who applied the plaster jacket testified at the trial, a month subsequently, that he was cured. Neither Dr. L. A. Stimson, nor Dr. Hamilton, nor Dr. Clymer could discover indications of its existence. It is quite evident that he did not have Pott's disease on the 4th of November, when I saw him, or at any subsequent period, and exceedingly probable that he had never had it.

He complained, however, of pain throughout the whole spine, and of excessive nervous irritability. He had had contractions of the muscles of the lower extremities, and on causing him to walk about the room it was evident that his limbs were stiff, and that he lifted his feet with difficulty. His gait was very different from that of a person suffering from locomotor ataxia. The feet were not raised from the ground with a jerk and put down with the two distinct

movements so characteristic of locomotor ataxia, but were moved as if they were weighted down with some heavy substance. The knee-tendon reflex was greatly exaggerated on both sides.

Up to this time no experiments had been made with the view of testing the sensibility of the lower extremities. These were now denuded of their clothing, and the patient was told to shut his eyes. The touch of a finger, the scratch of a pin, or a deep puncture with the blade of a pen-knife was equally unfelt in the right leg. On making the like experiments on the left leg, he complained of pain when the knife was stuck into it, and automatically carried his hand to the place which he supposed I had punctured, but, instead of touching the spot injured, he indicated the exactly corresponding situation on the other leg. Repeated experiments led to like results. He had sensibility in the left leg, but referred all impressions to the other side. Dr. Stimson assisted in verifying these results.

I came to the conclusion that the patient was suffering from antero-lateral or lateral sclerosis, with the implication of the posterior horns of gray matter, and probably of the membranes of the cord to a slight extent.

With the diagnosis, however, I have little to do at present, my intention being to restrict what I have to say to the crossed sensibility which the patient exhibited. To this condition the name allochiria (ἄλλος, χείρ) has been given by Professor Obersteiner,[1] of Vienna, who was the first, so far as I know, to call special attention to the phenomenon, though it had been incidentally alluded to by Leyden, and one or two others, as an occasional symptom of locomotor ataxia. A case following severe cranial injury has also been reported by Ferrier.[2]

Of Obersteiner's four cases, two were of locomotor ataxia, one was hysterical, and the other was the result of severe and direct injury of the spine. Death ensued in this last case, and, on post-mortem examination, it was found that there had been inflammation of the first, second, and third

[1] "On Allochiria, a Peculiar Sensory Disorder," *Brain*, July, 1881, p. 153.
[2] "Case of Allochiria," *Brain*, October, 1882, p. 389.

lumbar vertebræ, meningitis, and extensive transverse inflammation of the cord. The posterior columns, for a considerable distance above the seat of the injury, were in a state of sclerosis, and the posterior horns of gray matter in portions of the cervical enlargement were "transversely divided by a peculiar, structureless, transparent mass, intensely colored by carmine, and very similar to the mass which is found round the larger vessels in inflammatory processes in the cord."

I have quoted Obersteiner's own language because I think it is to such a lesion of the posterior horns of gray matter as he describes that the phenomenon of allochiria is to be ascribed. Neither he nor Ferrier offer any explanation of the mechanism of its production. On the contrary, they declare their inability to do so.

Certainly allochiria is not a usual symptom of sclerosis of the posterior columns of the spinal cord. I do not think it is ever met with in uncomplicated cases of this disease, nor do I think it is a possible condition in such instances. For the complete understanding of the subject, a few words relative to the anatomy and physiology of the cord are necessary.

The posterior tract of gray matter is probably the only channel by which sensory impressions reach the brain, the posterior columns having, in their normal condition, nothing whatever to do with the transmission of such impressions. But, before reaching the posterior horns, the posterior roots of the spinal nerves pass through the columns of Burdach, and, when these are the seat of inflammation, as they are in locomotor ataxia, disturbances of sensibility, such as hyperæsthesia, paræsthesia, and anæsthesia, are produced in the parts below by the pressure exerted upon these roots,

It is quite certain, as Brown-Séquard, Lockhart Clarke, Gerlach, and others claim, that there is an almost complete decussation of the sensory fibres within the gray matter—those from the right side of the body passing over to the left side of the cord, and *vice versâ*. We are taught these facts, not only by experimental physiology, but also by the instruction which we derive from the study of cases of dis-

ease or injury of the cord. Disregarding, as of no importance in the present connection, the fibres that do not decussate, we have in the accompanying diagram (Fig. 1)

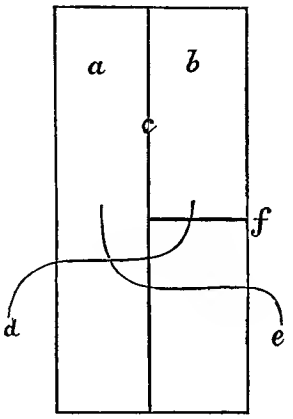


FIG. 1.

an explanation of the phenomena: *a*, the left posterior horn of gray matter; *b*, the right posterior horn; *c*, the commissure of gray fibres; *d*, sensory fibres coming from the left side of the body; *e*, sensory fibres coming from the right side. A lesion of the right posterior horn at *f* would produce anæsthesia of the left side of the body, and *vice versa.*

Now, in sclerosis of that portion of the posterior column called the column of Burdach, the lesion is almost always symmetrical, both sides being equally and correspondingly affected. As a consequence, we have in the latter stages more or less profound anæsthesia and retardation of the conveyance of sensory impressions in both lower extremities, and this not only from pressure exerted upon the posterior roots of the spinal nerves, but from an extension of the morbid process to both posterior horns of gray matter, exactly as would be the case if the line *f* in the diagram were prolonged so as to interfere with the nerve *e*. Allochiria is in such cases an impossibility, for all channels to the brain are closed, wholly or in part, and the patient either does not feel at all or feels imperfectly in the parts below.

But, in those cases of disease or injury of the posterior horns of gray matter, whether they be primarily involved or secondarily, as in locomotor ataxia in which allochiria exists, either the lesion must be unilateral, or, if both horns are involved, the lesions must be at different levels. In either case, as Dr. Morton suggested in a discussion in which our views of the subject were interchanged, allochiria must exist. The accompanying diagram (Fig. 2) will make this

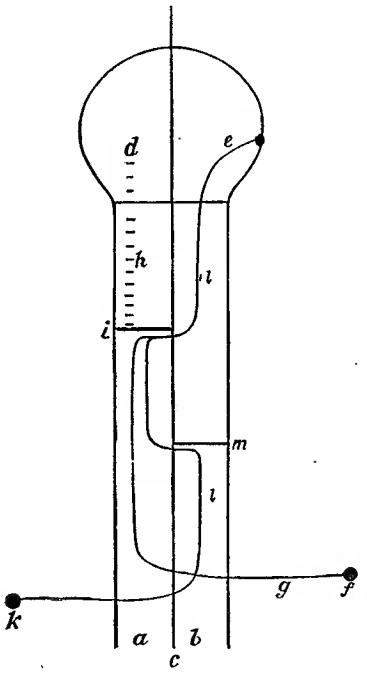


FIG. 2.

plain: Let *a* represent the left posterior horn of gray matter and *b* the right, *c* the gray commissure, *d* the left hemisphere, and *e* the right. A sensation starting at *f* in the right lower extremity would in the normal condition follow the fibres *g* and *h* to reach the cortex *d*, where it would be referred to its proper situation at *f*. But suppose there is a lesion in the left posterior horn at *i*; then the sensation would be directed through the gray commissural fibres to

the right posterior horn, and would reach the cortical centre in the right hemisphere, which is in relation with fibres coming from the left side of the body. The sensation, would, therefore, be referred to *k* through the fibres *l*, *l*. This would constitute the condition of allochiria, in which all impressions made, for instance, on the right side of the body, would be felt on the left, while those made on the left would be felt in their proper situations.

But suppose there is another lesion. If this is symmetrical with that on the right side at *i*, it is evident that no sensorial impressions from either side can reach the brain; there will be absolute anæsthesia in all parts below the lesion. Let us further suppose, however, that the other lesion is lower down, at *m*. Then impressions coming from *k* will be diverted to the left side on reaching the obstruction, and, arriving at *i*, will either be altogether arrested, leading to complete anæsthesia at *k*, or will be again diverted, and, reaching *e*, though with their strength greatly impaired, will be imperfectly felt at *k*. Such lesions explain those cases in which there is absolute anæsthesia on one side of the body, with sensation on the other side for impressions coming from both sides. They also show, as Obersteiner asserts, that anæsthesia is not a necessary concomitant of allochiria.

In the only case of allochiria in which a post-mortem examination has been made, and to which I have already alluded, Obersteiner found, among other abnormal conditions, disease of both posterior horns of gray matter. The morbid process was not continuous, as it is stated that it was not perceived in all the sections. It was situated at the narrowest part of the posterior horns, being so placed as to interrupt the decussation of all the nerve fibres, and hence to cause the transmission of sensory impressions upward in the side in which they entered—a condition which, equally with that I have described, would give rise to allochiria.

It is a well-known physiological fact that section of one lateral half of the spinal cord gives rise not only to anæsthesia of the parts below on the opposite side of the body,

but to hyperæsthesia of the parts below on the same side. This circumstance, which has not hitherto been explained, is, I think, satisfactorily accounted for by the theory I have proposed. For the parts below, corresponding to the cut half of the cord—for example, the right—not only remain in undisturbed relation with their proper cortical centre in the left hemisphere, but this latter receives also the sensory impressions coming from the left side. There will therefore be increased sensibility in the right side. Numerous facts in morbid anatomy and pathology could readily be brought forward in support of this view.

This explanation of the cause of crossed hyperæsthesia is different from the ingenious one of Ott,[1] though probably not irreconcilable with it.

Stated Meeting, Feb. 6, 1883.

Dr. S. N. LEO read a brief paper entitled "Presentation of patients trephined for epilepsy."

Henry S., peddler, aged forty years, native of Poland, single; had no less than fourteen characteristic epileptic fits within four hours, with continuous convulsive twitchings on the right side. But little could be gleaned from him or his previous history then, though he gave me to understand that he had terrible pain on the left side of his head, almost circumscribed to an angle between the squamous and coronal sutures, slightly above the former; and a friend volunteered the information that it was all due to a blow, which had been inflicted by a companion with a heavy stick, some four years prior, and which the sufferer claimed in his few lucid intervals, caused his trouble. European physicians of pronounced eminence said it was a singular but incurable case, and had prescribed a variety of treatment, which only afforded temporary relief. The pupil on the left side was dilated, while that on the right was contracted. There was no swelling or change over the supposed site of injury, excepting a slightly abraded surface about half an inch in circumference, uncovered by hair, and where, it was stoutly maintained, he had received the blow previously mentioned. There was ptosis of the left eyelid, loss of power in the left hand and leg, slight paralysis of vesical and

[1] *Journal of Physiology*, vol. ii, No. 2.

sphincter muscles, labored respiration, and with great difficulty could food be swallowed. The heart's action was irregular, and as the same state of affairs had continued, on and off, without any amelioration for nearly three days, I trephined within the ensuing twenty-four hours, under the most adverse circumstances. An anæsthetic was administered with caution, after which a small trephine was employed.

The operation lasted thirty minutes, the wound being carefully dressed ; the man had but one attack that evening.

The next day he felt much better ; *no fits.* On the third day following the operation had but a slight attack, sight improved, regained control of all his muscles, spoke rationally, pain in the head all gone, and from that time on continued to mend, and with the exception of a troublesome facial neuralgia, extending over the whole right side, did well, and he eventually resumed his business.

Lawrence W., aged twenty-five ; married ; U. S.; cigar-maker. Admitted August 5, 1882, to Charity Hospital.

When about thirteen years old was struck in the back of the head with a brick ; he fell upon the car-track stunned, and remained insensible for some time. Epileptic attacks occurred when he was about nineteen.

Patient has no premonition ; utters a prolonged cry ; head turns to the right, and backward. Convulsion is limited to right side of face and neck and right arm. Is of short duration. Bites tongue and cheek. Recovers with a start as if surprised. All his attacks have been the same in character.

First day of admission, Saturday, had twenty-six fits in about twelve hours ; while in the reception office had four. Thirty-four fits Sunday night. Monday, had sixty-seven fits. Tuesday, total number of fits, one hundred and ninety. Wednesday, total for the day, one hundred and forty. Thursday, frequency of fits slightly abated ; slept very little ; always waking in a convulsion ; cried repeatedly. Head carefully examined at seat of injury, where he now complained of pain ; some roughening was found, and it was determined to trephine, as there were concomitant symptoms of compression (most probably from effusion).

At this time patient could not repeat any word distinctly, except monosyllables, and could not swallow but very little. The greater part of food taken into the mouth was regurgitated ; bowels moved regularly every day ; urine passed, without exception, examined and found normal. (Alkaline reaction.)

Friday, 11th.—Up to two o'clock had nineteen fits.

In the presence of Dr. Seaman, Chief of Staff, and several other physicians, I proceeded to operate, Dr. O'Brien having etherized the patient, when I raised a V-shaped flap, removed a disc about one inch in diameter at a point one and one half inches backward and upward from the right mastoid process, and over the squamous ridge of the temporal bone. There was but a moderate amount of hemorrhage easily controlled; serous exudation external to the dura mater was noticed, and permitted to flow off. Abundant evidence of an organized inflammatory process, that had doubtless given rise to the chain of symptoms, was observed, and which, if unchecked, unquestionably would have gone from bad to worse. Operation lasted forty minutes; patient shortly recovered from the anæsthetic, and conversed; dressings of carbolized cloths applied; clothes changed, and the man fell into a slumber, which lasted nearly four hours; awoke in another convulsion; during the night slept considerably; had seven more attacks; total eight.

Sunday, 13th—Treatment continued; reapplied dressings; complains of no headache or untoward symptom; bowels moved. Temperature 99°, pulse 96, six P.M. Twitches in face same side as operation. *Had no fits to-day.*

Friday, 25th—Is very well to-day, except slight diarrhœa. From this time on the patient has made good progress, and for some months has resumed his occupation of cigar-making.

Judging by these two cases, it would seem that in subjects who suffer from numerous severe epileptic attacks or convulsions, where there is a direct irritation of the brain, depressed fracture, intra-cranial effusion, or other causative influence producing a compression, they should be trephined, especially if, after an extended trial of medication, no appreciable benefit is derived.

Dr. LEALE inquired how many attacks had occurred subsequent to the two instances of operation reported.

Dr. LEO replied that in the first instance two years had elapsed since attacks, and in the second three months.

Dr. L. C. GRAY thought that though the attacks had ceased the epileptic habit might yet remain.

Dr. PUTZEL asked how Dr. Leo accounted for the serous fluid that he reported as existing in each instance between the bone and the dura mater.

Dr. LEO thought that it probably was an inflammatory product; in the first case it escaped with a gush as soon as the bone had been perforated; it was yellow in color, and certainly exterior to the dura mater.

Dr. PUTZEL thought that serous fluid in this locality was very rare,

Dr. GRAEME HAMMOND referred to a case operated upon by his father, Dr. Wm. A. Hammond, in which no recurrence of the attack had appeared up to the present time, a period of five or six years.

Dr. LEALE related an instance where trephining had been performed in the case of a young girl who had been subject to frequent attacks of "fits," and in which the immunity to attacks had now lasted to his knowledge for one year.

Dr. MORTON remarked that the question of the length of time during which the patient enjoyed immunity to the attacks after the operation, was an important point in the reports of these cases. For instance, he recalled assisting Dr. Hammond some years since in an operation upon a confirmed epileptic where attacks were evidently due to traumatism. [The patient had been struck on the head by an ice-pick.] The cutaneous cicatrix offered a clear guide to the locality for operating. The button of bone removed showed merely a slight thickening, perfectly smooth on its under surface, while the subjacent dura mater appeared normal. The attacks, frequent before the operation, ceased entirely after it, and not having recurred at the end of two years, the patient was considered cured. His physician, however, had lately reported that attacks had again made their appearance, though greatly diminished in frequency.

Dr. Morton also referred to a case where he had trephined for epileptoid convulsions occurring in a recent injury to the skull from the kick of a horse. The dura was found to be ruptured and the brain injured. The convulsions ceased after the operation, but the patient died some weeks subsequently from an extensive abscess of the brain.

The discussion of a definition of insanity was postponed to a subsequent meeting.

The Society then adjourned.

Stated Meeting, March 6, 1883,

Dr. WM. A. HAMMOND read a paper upon "Katatonia."

Dr. SPITZKA read a paper on "A Classification of insanity."

Nominations of officers for the ensuing year were as follows:

For President, William J. Morton, M.D., and L. C. Gray, M.D.; for Vice-President, V. P. Gibney, M.D., and L. Weber, M.D.; for 2d Vice-President, W. H. Farrington, M.D.; for Recording Secretary, C. L. Dana, M.D.; for Corresponding Secretary, M. Putnam Jacobi, M.D.; for Treasurer, E. C. Harwood, M.D.

The meeting then adjourned.

Annual Meeting, April 3, 1883.

The election of officers for the ensuing year was the only business transacted. The following officers were elected: President, William J. Morton, M.D.; 1st Vice-President, L. Weber, M.D.; 2d Vice-President, W. H. Farrington, M.D.; Recording Secretary, M. Josiah Roberts, M.D.; Corresponding Secretary, M. P. Jacobi, M.D.; Treasurer, E. C. Harwood, M.D.; Councillors, Wm. A. Hammond, M.D., E. C. Seguin, M.D., T. A. McBride, M.D., R. Birdsall, M.D., G. W. Jacoby, M.D.

Dr. MORTON presented a resolution to the effect that an official report of the transactions of the Society at its monthly meetings should be made and furnished to medical journals. This resolution was seconded and referred to the Council.

The Society then adjourned.